RIGHT VENTRICULAR FUNCTION IN INFERIOR WALL MYOCARDIAL INFARCTION – CLINICAL AND ECHOCARDIOGRAPHIC CORRELATION

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CERTIFICATE

This is to certify that this dissertation titled **"RIGHT VENTRICULAR FUNCTION IN INFERIOR WALL MYOCARDIAL INFARCTION – CLINICAL AND ECHOCARDIOGRAPHIC CORRELATION"** submitted by **Dr. Sony Manuel.M** to the faculty of General Medicine, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch I (General Medicine) is a bonafide research work carried out by him under our direct supervision and guidance.

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ABBREVIATIONS AND ACRONYMS

IW - Inferior wall.

RV-Right ventricle.

LV-Left Ventricle.

MI-Myocardial Infarction.

ACS-Acute Coronary Syndrome.

CAD-Coronary Artery Disease.

AS-Aortic Stenosis.

AR-Aortic Regurgitation.

MINCA- Myocardial Infarction with Normal Coronary Arteries.

RCA – Right Coronary Artery.

CK – Creatine Kinase.

ECG-Electrocardiography.

TAM-Tricuspid Annular Motion.

TAFC-Tricuspid Annular Fractional Shortening.

TAPSE-Tricuspid Annular Peak Systolic Excursion.

TDI - Tissue Doppler imaging.

IVS – Interventricular septum.

RV WT- Right ventricle wall thickness.

RV WM – Right ventricle Wall Motion.

RV ESA – Right ventricle end systolic area.

RVEDA – Right ventricle end diastolic area.

RV FAC - Right ventricle fractional area change.

IVCT – Isovolumetric contraction time.

IVRT – Isovolumetric relaxation time.

EF – Ejection fraction.

ET- Ejection time.

MPI – Myocardial Performance Index.

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INTRODUCTION

Right ventricular (RV) function has not been widely studied after a myocardial infarction (MI) unlike LV function[1]. There is RV involvement in more than 1/3rd of patients with acute IWMI. RV involvement has been reported to be an independent predictor of major complications and in-hospital mortality after acute inferior MI.

ST-segment elevation in the right precordial lead, V_4R, is one of the most reliable electrocardiographic signs of acute RV infarction . A hypokinetic or akinetic segment of the right ventricle observed by echocardiography also could be used to detect RV dysfunction after RV infarction. However, because of the complex shape, evaluation of RV function by echocardiography has been considered difficult. However, a previous study has used tricuspid annular motion to assess RV function. Myocardial velocity determined by Doppler tissue imaging is a new technique that has been used recently to analyse left ventricular function. The development of DTI opens up the possibility of also assessing RV function. However, this technique has not been used to assess RV function after myocardial infarction . With the use of both the tricuspid annular motion and tricuspid annular velocity, the purpose of the current work is to study RV function in association with an acute first IWMI.

REVIEW OF LITERATURE

BACKGROUND

Involvement of RV is present in approximately 50 % of patients with acute inferior myocardial infarction and may result in hemodynamically compromised situation with a poor clinical outcome[(2)(70)]. Acute right coronary artery occlusion proximal to the RV branch often results in RV free wall dysfunction. Patients with RV myocardial infarction often respond to volume treatment and early reperfusion enhances the recovery of RV performance and significantly improves the clinical outcome and improves survival . Interest in recognizing right ventricular infarction non invasively has grown because of the therapeutic implications of distinguishing patients with right ventricular dysfunction from others. Patients with RVI have much higher rates of significant hypotension, bradycardia requiring pacing support, and in-hospital mortality than isolated inferior infarctions.

Myocardial infarction typically progresses through several stages:

ACUTE : Encompassing the first few hours to 7 days following infarct onset.

HEALING : Occurring during days 7 to 28.

HEALED : Beginning on day 29 and continuing thereafter.

Revised Definition of Myocardial Infarction [3]

Criteria for acute, evolving or recent MI

Either of the following criteria satisfies the diagnosis for acute, evolving or recent MI:

(1) Typical rise and/or fall of biochemical markers of myocardial necrosis with at least one of the following:

(a) ischemic symptoms.

(b)development of pathological Q waves in the ECG.

(c)ECG changes indicative of ischemia.

(d)Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

(2) Pathological findings of an acute MI.

Criteria for healing or healed MI

Any one of the following criteria satisfies the diagnosis for healing or healed MI.

(1) Development of new pathological Q waves in serial ECGs .The patient may not remember previous symptoms .Biochemical markers of myocardial necrosis may have normalised depending on the length of time that has passed since the infarction developed.

(2) Pathological findings of a healing / healed infarction.

Clinical Classification of Different Types of Myocardial Infarction[3]

Type 1

Spontaneous myocardial infarction related to ischemia due to a primary coronary event.

Type 2

Myocardial infarction secondary to ischemia due to either increased oxygen demand or decreased supply.

Type 3

Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, accompanied by presumably new ST elevation, or new LBBB orevidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers inthe blood.

Type 4a

Myocardial infarction associated with PCI.

Type 4b

MI associated with stent thrombosis as documented by angiography or at autopsy

Type 5

Myocardial infarction associated with CABG

Etiology[2]

(1) Coronary Atherosclerosis (almost all cases).

(2)Non atherogenic causes :

- ❖ CAD other than Atherosclerosis
  - Arteritis.
  - Trauma to coronary arteries.
  - Coronary mural thickening with metabolic disease or intimal proliferative disease.
  - Luminal narrowing : spasm, dissection.
- ❖ Emboli to Coronary arteries.
- ❖ Congenital coronary artery anomalies.
- ❖ Myocardial oxygen demand-supply disproportion.

  Aortic Stenosis, Aortic Regurgitation, Carbon monoxide poisoning, Prolonged hypotension, Takotsubo cardiomyopathy.
- ❖ Hematological (in situ thrombosis) :Disseminated Intravascular Coagulation, Polycythemia vera, Thrombocytosis.
- ❖ Miscellaneous : Cocaine, Myocardial contusion, MINCA, Complication of cardiac catheterisation.

Pathophysiology[4]

In majority of patients, acute MI occurs as a result of atherosclerosis. STEMI occurs when a coronary artery thrombus develops rapidly at a site of vascular injury. This injury is produced or facilitated by factors such as cigarette smoking, hypertension, and lipid accumulation. A mural thrombus forms at the site of plaque disruption, and the involved coronary artery becomes occluded. Histologic studies indicate that the coronary plaques prone to disruption are those with a rich lipid core and a thin fibrous cap[4]**(Vulnerable Plaque).**

The amount of myocardial damage caused by coronary occlusion depends on (1) the territory supplied by the affected vessel,

(2) whether or not the vessel becomes totally occluded,

(3) the duration of coronary occlusion,

(4) the quantity of blood supplied by collateral vessels to the affected tissue,

(5) the demand for oxygen of the myocardium whose blood supply has been suddenly limited,

(6) native factors that can produce early spontaneous lysis of the occlusive thrombus, and

(7) the adequacy of myocardial perfusion in the infarct zone when flow is restored in the occluded epicardial coronary artery.

Risk Factors for atherosclerosis[2]

Conventional.. age, sex, smoking, hypertension, hyperlipidemia, insulin resistance and diabetes, depression and mental stress.

Novel Risk factors.

- Markers of inflammation like
  hsCRP,
  IL-6,
  sICAM-1,
  P selectin or the mediator CD-40 ligand,
  myeloperoxidase,
  lipoprotein-associated phospholipase A_2,
  pregnancy associated plasma protein A.
- Lipoprotein(a),
- Homocysteine,
- Markers of fibrinolytic and hemostatic function such as fibrinogen,
  D-dimer,
  imbalance between tissue plasminogen activator and plasminogen activator inhibitor 1 antigens.

Clinical features of MI

Symptoms[2][4]

Most common presenting complaint is central,squeezing type of chest pain which is more severe, occurs even at rest and usually lasting more than 30 minutes.It commonly radiates down the ulnar aspect of the left arm: may radiate as high as the occipital area but not below the umbilicus. It is often accompanied by weakness, sweating, nausea, vomiting and a sense of impending doom. In up to 50% cases , a precipitating factor appears to be present before STEMI such as vigorous physical exercise, emotional stress, a medical or surgical illness .The time of onset of STEMI has a pronounced circadian periodicity with peak incidence between 6 am and noon as the early morning hours are associated with rises in plasma catecholamines and cortisol and increases in platelet aggregability .However pain is not uniformly present in all patients with STEMI [69].

Painless STEMI may be seen in diabetics, elderly, women, recipients of heart transplants and post operative patients [5] . These patients usually present with features of left ventricular failure like sudden onset breathlessness.

Other less common presentations include syncope, a confusional state, the appearance of an arrhythmia, evidence of peripheral embolism or merely an unexplained drop in arterial pressure.

Physical Findings[4]

Most patients are anxious and restless.Pallor associated with perspiration and coolness of the extremities occurs commonly. The combination of substernal chest pain persisting for >20min[61] and diaphoresis strongly suggests STEMI. Although many patients have a normal pulse rate and blood pressure within the first hour of STEMI, about one-fourth of patients with anterior infarction have manifestations of sympathetic nervous system hyperactivity (tachycardia and/or hypertension) and up to one-half with inferior infarction show evidence of parasympathetic hyperactivity (bradycardia and/or hypotension). The carotid pulse is often decreased in volume, reflecting reduced stroke volume. Temperature elevations up to 38°C may be observed during the first week after STEMI. The arterial pressure is variable; in most patients with transmural infarction, systolic pressure declines by approximately 10–15 mmHg from the preinfarction state. The precordium is usually quiet, and the apical impulse may be difficult to palpate. In patients with anterior wall infarction, an abnormal systolic pulsation caused by dyskinetic bulging of infarcted myocardium may develop in the periapical area within the first days of the illness and then may resolve. Other physical signs of ventricular dysfunction include fourth and third heart sounds, decreased intensity of the first heart sound, and paradoxical

splitting of the second heart sound. A transient midsystolic or late systolic apical systolic murmur due to dysfunction of the mitral valve apparatus may be present. A pericardial friction rub is heard in many patients with transmural STEMI at some time in the course of the disease commonly on the second or third day[2].

Hemodynamic Classification of Patients with Acute MI

(A)Based on Clinical Examination [6] (Killip's Classification)		(B)Based on Invasive Monitoring[7] (Forrester Classification)	
Class	**Definition**	**Subset**	**Definition**
I	**Rales and S_3 absent**	**I**	**Normal hemodynamics** **PCWP<18 ; CI>2.2**
II	**Crackles , S_3 gallop,** **Elevated JVP**	**II**	**Pulmonary Congestion** **PCWP>18 , CI>2.2**
III	**Frank Pulmonary Edema**	**III**	**Peripheral Hypoperfusion** **PCWP<18 ; CI<2.2**
IV	**Shock**	**IV**	**Pulmonary Congestion and Peripheral Hypoperfusion** **PCWP>18 ; CI<2.2**

PCWP = pulmonary capillary wedge pressure ; **CI**=cardiac index

RIGHT VENTRICULAR INFARCTION (RVI)

Approximately 50% of patients with IWMI have some involvement of the RV[(2)].Among these patients, RVMI occurs exclusively in those with transmural infarction of the inferoposterior wall and the posterior portion of the septum. Patients with inferior MI who also have RV myocardial involvement are at increased risk of death, shock and arrhythmias **(Kukla *et al.* 2006[(8)],Khan S *et al*;2004[(9)])**. This increased risk is related to the presence of RV myocardial involvement itself rather than the extent of LV myocardial damage[**Mehta *et al* 2001[(10)]**] . IWMI patients with RVI comprises a high-risk subset of patients with a mortality rate as high as 25% to 30%, as opposed to an overall mortality rate of approximately 6% of patients with inferior MI without RVI. Right ventricular infarction is an independent risk factor for increased mortality even in these days of primary PCI [**Abid R. Assali *et al*.2007 [(11)]**] Right ventricular function is an independent predictor of death and development of heart failure in patients with LV dysfunction after MI **[Zornoff *et al*. 2002[(12)]]**.

Anavekar, *et al* 2008[(13)] in their study observed that RV function was a predictor not just of death and hospitalization for HF, but also of stroke and sudden death.The mechanism for this relation is unknown.

Right ventricular function may be a sensitive measure of left atrial pressure, and decreased RV function may reflect chronically increased left atrial pressures, associated with larger atria and atrial stasis, risk factors for atrial thrombus formation and stroke.

Isolated infarction of the RV is seen in 3-5% of autopsy-proven cases of MI. The less frequency of involvement of RV can be explained by the lesser oxygen demands of the RV, rich intercoronary collateral system of the RV, thinness of the RV walls allowing it to derive some nutrition from the RV cavity [**Goldstein 2002]**[(14)]. So RV can sustain long periods of ischemia but still demonstrate excellent recovery of contractile function after reperfusion. Although there are potentially life threatening acute hemodynamic and clinical consequences in some, most patients with RV dysfunction after MI have spontaneous recovery of RV function, leading some clinicians to believe that the term "right ventricular infarction" is a misnomer and represents viable but "stunned" myocardium [**Haji *et al.* 2000** [(15)]]. **Dimitrios *et al.* 2004** [(16)] observed that the systolic and diastolic function which was seriously impaired during the MI involving RV showed significant improvement after 3 months of the acute event; this significant improvement during the follow-up may represent a possible recovery of stunned myocardium.

Ramzy *et al*.2008 [(17)] in their study observed that in IWMI, RV segmental and global functions are acutely impaired and recover in 87%

of patients following thrombolysis; In the absence of clear evidence for RV infarction, the disturbances in the remaining 13% may represent stunned myocardium that may demonstrate delayed recovery.

The posterior descending branch of the right coronary artery usually supplies the inferior and posterior walls of the right ventricle. The marginal branches of the right coronary artery supply the lateral wall of the right ventricle. The anterior wall of the right ventricle has a dual blood supply: the conus branch of the right coronary artery and the moderator branch artery, which courses from the left anterior descending artery **(Forman *et al,* 1984)** [(18)].Interestingly, RVI noted at necropsy usually involves the posterior septum and posterior wall rather than the right free wall. The relative sparing of the RV anterior wall apparently arises from a high degree of collateralisation. This collateral blood flow is thought to be derived from the thebesian veins and diffusion of oxygen directly from the ventricular cavity. A direct correlation exists between the anatomic site of RCA occlusion and the extent of RVI. Studies have demonstrated that more proximal RCA occlusion results in larger RVMI. **(Garty *et al*. 1984)**[(19)].

Acute RCA occlusion proximal to the RV branches results in RV free wall dysfunction, exerting mechanically disadvantageous effects on biventricular performance. Depressed RV systolic function decreases

transpulmonary delivery of left ventricular (LV) preload , resulting in diminished cardiac output. The ischemic right ventricle is stiff, dilated, and volume dependent, resulting in pan diastolic RV dysfunction and septally mediated alterations in LV compliance, which are exacerbated by elevated intra pericardial pressure. Under these conditions, RV pressure generation and output are dependent on LV septal contractile contributions, governed by both primary septal contraction and paradoxical septal motion. When the culprit coronary lesion is distal to the right atrial (RA) branches, augmented RA contractility enhances RV performance and optimises cardiac output. Conversely, more proximal occlusions result in ischemic depression of RA contractility, which impairs RV filling and performance, resulting in more severe hemodynamic compromise **(Goldstein 2002)**[14].

On occasion, the right ventricle can be subjected to infarction from occlusion of the left circumflex coronary artery **(Giannitsis *et al*, 1997)**[20] . Because the RV is considered a low-pressure volume pump, its contractility is highly dependent on diastolic pressure. Hence, when contractility and associated diastolic function are impaired attendant to RVI, the RV diastolic pressure increases substantially and systolic pressure decreases. In such a scenario, concomitant LV dysfunction, with increase in RV afterload, is possible. In such a setting, RV output can

decrease dramatically, and the only driving force remaining is elevated right atrial pressure. In such a circumstance, the RV serves as a poorly functioning conduit between the right atrium and the pulmonary artery. Elevation of right atrial pressure secondary to RVI has been noted to serve as a stimulus for secretion of atrial natriuretic factor. Increased levels of this polypeptide can be detrimental to normal left ventricular filling pressures. This occurs by virtue of the potent vasodilating, natriuretic, diuretic, and aldosterone-inhibiting properties of atrial natriuretic factor. Inappropriately elevated levels of atrial natriuretic factor may worsen the clinical syndrome of right ventricular infarction **(Haupt *et al,* 1983)**[21] .The potential hemodynamic derangements associated with RVI render the afflicted patient unusually sensitive to diminished preload and loss of atrioventricular synchrony. These two circumstances can result in a severe decrease in right and secondarily LV output**(Hirsowitz *etal* 1984;Hurst1998;Iqbal and Liebson1981)** [22,23,24]**.** Early thrombolysis or mechanical reperfusion of an occluded coronary artery resulting in RVI is associated with prompt reduction in right atrial pressure. This is extremely important because persistently elevated right atrial pressure has been associated with increased in-hospital mortality rate when associated with MI. The extent of RVI varies greatly and is dependent on the site of occlusion of the RV arterial supply. If occlusion occurs before the RV marginal branches, and collateral blood flow from

the left anterior descending coronary artery is absent, then the size of infarction generally is greater. Extent of infarction depends somewhat on flow through the thebesian veins **(Kinn *et al*, 1995)** [(25)] . In general, any major reduction in blood supply to the right ventricular free wall portends an adverse prognosis in association with this disorder.

The classic clinical triad of right ventricular infarction includes distended neck veins, clear lung fields, and hypotension **(Mavric et al, 1990)** [(26)]**.**

Jacobs.*et al* ,2003[(27)]found that patients with predominant RV shock were younger than patients with LV shock, although CAD risk factor profile, with the exception of hyperlipidemia, was similar between the two groups.There was a lower incidence of previous MI for patients with predominant RV shock as the patients with predominant RV shock were more likely to have single- and double-vessel disease and less likely to have triple-vessel disease than patients with LV shock.

Rare manifestations include RV third and fourth heart sounds, which are typically audible at the left lower sternal border and increase with inspiration.A subtle clue to the presence of hemodynamically significant right ventricular infarction is a marked sensitivity to preload-reducing agents such as nitrates, morphine, or diuretics **(Mittal, 1994)**[(28)].Other

presentations include high-grade atrioventricular block, tricuspid regurgitation, cardiogenic shock, RV free wall rupture, and cardiac tamponade. Unexplained hypoxia despite administration of 100% oxygen in a case of RVMI can be due to right-to-left shunting at the atrial level in the presence of RV failure and increased right atrial pressure must be considered. The mechanism for right-to-left shunting in the absence of increased pulmonary arterial pressure resides in patency of the foramen ovale in association with poor right ventricular compliance and increased right atrial filling pressures. Patients with extensive right ventricular necrosis are at risk for RV catheter related perforation, and passage of a floating balloon catheter or pacemaker must always be performed with great care in such a setting. In the appropriate clinical setting, a diagnosis of RVI can be made using non invasive techniques, or the patient may require RV catheterisation and hemodynamic monitoring.

Echocardiography is useful as a modality to rule out pericardial disease and tamponade, which are the major differential diagnoses in the setting of a RVI. RV dilatation, abnormal RV wall motion, paradoxical motion of the interventricular septum, and tricuspid regurgitation are echocardiographic features of RVI. Echocardiogram can detect shunting through a patent foramen ovale. Echocardiogram has an 82% sensitivity and 93% specificity in detecting RVI when right ventricular scintigraphy

is used as the comparative standard **(Singhal *et al*, 1984)**[29].In the vast majority of patients with RVI, the wall motion abnormalities initially manifest on echocardiography reverse within 3 months **(Strauss *et al*, 1980)** [30] .The recent utilisation of tissue doppler in echocardiography has also increased, providing another means to detect RVI. A decrease in the systolic velocity at the tricuspid annulus not only allows for diagnosis of RVI but also suggests worse outcome **(Hisham, 2005)**[31].

Another echocardiographically obtained value that can aid in diagnosis of RVI is the myocardial performance index (MPI). MPI is derived from the sum of the isovolumic relaxation and contraction time divided by the ejection fraction. An abnormally elevated MPI of $\geq$ 0.30 suggests the presence of a right ventricular infarction **(Chockalingam,2004)**[32].

Gated equilibrium radionuclide angiography and technetium ^{99}m pyrophosphate scintigraphy are useful in diagnosing right ventricular infarction noninvasively **(Sugimoto *et al*, 1996)**[33].

In the case of radionuclide angiography, the RV is demonstrated to be enlarged and poorly contractile, with a reduced ejection fraction. When technetium 99m pyrophosphate is employed, the RV free wall is "hot" indicating significant infarction.

On hemodynamic monitoring, disproportionate elevation of right-sided filling pressures compared with left-sided hemodynamics represents the hallmark of RVI. Hemodynamic measurements in patients with significant RVMI demonstrate elevation of the right atrial pressure, usually more than 10 mmHg, and often show a right atrial pressure / pulmonary artery wedge pressure ratio of 0.8 or more. However, in cases with significant LV dysfunction and increased wedge pressure, this ratio may be lower and does not exclude the presence of significant RV involvement.

ELECTROCARDIOGRAPHY IN MYOCARDIAL INFARCTION

The inferior wall of the left ventricular cone is oriented to leads II,III and AVF. IWMI will therefore be reflected by the appearance of the classic features of hyperacute, fully evolved and chronic stabilised phase in these leads. Results that indicate high probability of MI are ST-segment elevation greater than 1 mm in 2 anatomically contiguous leads or the presence of new Q waves.

Localization of MI based on distribution of ECG abnormalities is as follows: Inferior wall - II, III, aVF, Lateral wall - I, aVL, V_4 through V_6, Anteroseptal - V_1 through V_3, Anterolateral - V_1 through V_6, Right ventricular - RV_4, RV_5, Posterior wall - R/S ratio >1 in V_1 and V_2; T-wave changes (ie, upright) in V_1, V_8, and V_9.

All patients with IWMI should have a right-sided ECG. ST-segment elevation in lead V4R is the single most powerful predictor of RV involvement, identifying a high-risk subset of patients in the setting of inferior wall myocardial infarction **(Robalino *et al*, 1990)** [34].

The presence of precordial ST-segment depression in acute IWMI is reported to be an ECG sign associated with larger infarct size extending to posterior segments and a higher rate of in-hospital complications. ST-segment elevation in V1 on admission indicates not only a RCA lesion but also a larger LV infarct size extending to the posterior segments because of a higher incidence of proximal lesions.

Among the patients with RVI, a higher incidence of multivessel disease was observed in patients with V1 $\geq$V4R compared to those with V1 < V4R. **[Yoshiaki Tsuka *et al.* 2001][35].**

The ST-segment elevation is transient, disappearing in less than 10 hours following its onset in half of patients.

Sensitivity and Specificity of more than 1 mm of ST-Segment Elevation in V_1, V_3R, and V_4R (Roth *et al*, 1990)[36] .

Leads	Sensitivity (%)	Specificity (%)
V_1	28	92
V_3R	69	97
V_4R	93	95

Isolated RVMI is extremely rare and may be interpreted erroneously as LV anteroseptal infarction on ECG because of ST-segment elevation in leads V_1-V_4 **(Schuler *et al*. 1984; Sharpe *et al*. 1978)**[37][38]. Some have suggested that the differential diagnosis between the two abnormalities can be distinguished by using vectorial analysis. The mean ST-segment vector in RVMI usually is directed anteriorly and to the right (>100°). In an anteroseptal LV infarct, the mean ST-segment vector is oriented leftward between -30° and -90° thus, analysis of the frontal and horizontal plane axis of the mean ST-segment vector can distinguish electrocardiographically between myocardial infarction at these 2 sites[70].

Serum Cardiac markers :

Myocardial cell death can be recognized by the appearance in the blood of different proteins released into the circulation from the damaged myocytes : myoglobin, cardiac troponin T and I , CK, Lactate Dehydrogenase, as well as many others . MI is diagnosed when blood levels of sensitive and specific biomarkers such as cardiac troponin or CK-MB are increased in the clinical setting of acute ischaemia.

The preferred biomarker for myocardial necrosis is cardiac troponin (I or T), which has nearly absolute myocardial tissue specificity as well as high clinical sensitivity, thereby reflecting even microscopic zones of myocardial necrosis . An increased value for cardiac troponin

(and CK) is defined as a measurement exceeding the 99th percentile of a normal reference population (URL = upper reference limit). Detection of a rise and/or fall of the measurements is essential to the diagnosis of acute MI. Blood samples for the measurement of troponin/CK should be drawn on first assessment and 6–9 h later . An occasional patient may require an additional sample between 12 and 24 h if the earlier measurements were not elevated and the clinical suspicion of MI is high . To establish the diagnosis of MI , one elevated value above the decision level is required. Since cTnT and cTnI are not normally detectable in the blood of healthy individuals but may increase after STEMI to levels >20 times higher than the URL , the measurement of cTnT or cTnI is of considerable diagnostic usefulness. Levels of cTnI and cTnT may remain elevated for 7–14 days after STEMI.

If troponin assays are not available, the best alternative is CKMB (measured by mass assay). Gender-specific values should be employed.

Creatine kinase (CK) rises within 4–8 h and generally returns to normal by 48–72 h. An important drawback of total CK measurement is its lack of specificity for STEMI, as CK may be elevated with skeletal muscle disease or trauma, including intramuscular injection. The MB isoenzyme of CK has the advantage over total CK that it is not present in

significant concentrations in extracardiac tissue and therefore is considerably more specific.

A ratio (relative index) of CKMB mass : CK activity >2.5 suggests but is not diagnostic of a myocardial rather than a skeletal muscle source for the CKMB elevation.

Normal levels of CK total in males : 51-294 U/L ; females: 39-238 U/L . Normal levels of CK-MB is < 12 IU/L if total CK is < 400IU/L and <3.5% if total CK is >400 IU/L.

Reinfarction : Traditionally, CKMB has been used to detect reinfarction. However, recent data suggest that troponin values provide similar information **(Apple FS *et al*.2005)** [39]. In patients where recurrent myocardial infarction is suspected from clinical signs or symptoms following the initial infarction, an immediate measurement of the employed cardiac marker is recommended . A second sample should be obtained 3–6 hours later. Recurrent infarction is diagnosed if there is a ≥20% increase of the value in the second sample.

ECHOCARDIOGRAM

It is one of the most important non invasive technique for cardiovascular diagnosis that gives reliable information together with patient safety .The echo sounding is something that the man has learnt from certain animals and birds which use this technique for distance perception. In 1920s,man used it for oceanographic studies and for submarine detection. Todays ultrasound diagnosis has its roots in the Navy Sonar,where it was useful in detecting objects and measuring distance in water.

In 1950s,Keidel first used ultrasound to examine the heart. In mid 1950s,Elder and Hertz pioneered the use of pulsed ultrasonic techniques to study the anatomy of the heart. In 1957, Holmes popularised the echocardiography by extensively using it in the study of cases of Mitral Stenosis . Ebina and others in 1960s brought in the two dimensional echocardiography .Only in the 1980,emerged the Doppler device due to investigations of Baker,a technique that added sound to the echocardiographic picture.The next in the evolutionary spectrum of echocardiography is the digital ultrasound which enhances the echo images for that applications as quantitative analysis and tissue characterization.TEE has markedly improved resolution of 2 D echo images .

Tissue Doppler imaging (TDI) is a new technology, which has been proposed for quantification of regional systolic and also diastolic regional function[67][71]. Routine Doppler imaging typically targets blood flow and hence the receiver characteristics, including the frequency filters that determine the range of velocities to be interrogated, are set to maximise the shifts anticipated with moving blood and to exclude the velocity shifts that would be seen with slower moving structures. Because red blood cells are relatively weak reflectors and tissue is a fairly intense reflector, filters are also adjusted to exclude highly reflective objects and to maximize less reflective objects when using conventional Doppler . TDI uses the same principles; however, the target is tissue rather than red blood cells. For this purpose, filters are set to parameters opposite those needed to accurately detect red blood cell motion. Because tissue has a greater reflectivity and slower motion, instrumentation filters are set to exclude high velocities and low-intensity reflectors. With this technique, either the myocardium or fibrous skeleton of the heart can be targeted and weaker reflections from the higher velocity blood cells relatively excluded. Myocardial velocity determined by DTI is a new technique that has been used recently to analyse LV function. The development of DTI opens up the possibility of assessing RV function. Detection of myocardial ischemia by visual assessment of wall motion is fraught with

variability and low reproducibility. Wall motion can be quantified by DTI. Low systolic tissue velocities correlate with angiographic or echocardiographic wall motion abnormality.

Tissue velocities decrease with reduced regional perfusion, recover on reperfusion, and differentiate between transmural and nontransmural MI [(68)]. Improvement in the ultrasound transducer technology created the harmonic transducer,allowing real time three dimensional echocardiography.

ECHOCARDIOGRAPHIC VIEWS OF THE RIGHT HEART [(40),(41)]

The normal RV is a complex crescent-shaped structure wrapped around the LV and is incompletely visualised in any single 2D view. Thus, accurate assessment of RV morphology and function requires integration of multiple views, including parasternal long- and short-axis, RV inflow, apical 4-chamber, and subcostal. Although multiple methods for quantitative echocardiographic RV assessment have been described, in clinical practice assessment of RV structure and function remains mostly qualitative. Compared with the LV, the RV is a thin-walled structure under normal conditions. The normal RV is accustomed to a low pulmonary resistance and, hence, low afterload ; thus, normal RV pressure is low and RV compliance high. The RV is therefore sensitive to changes in afterload, and alterations in RV size and function are

indicators of increased pulmonary vascular resistance and load transmitted from the left-sided chambers. Elevations in RV afterload in adults are manifested acutely by RV dilatation and chronically by concentric RV hypertrophy. In addition, intrinsic RV abnormalities, such as infarction or RV dysplasia can cause RV dilatation or reduced RV wall thickness. Thus, assessment of RV size and wall thickness is integral to the assessment of RV function.

(1)RV free wall thickness

RV free wall thickness, normally less than 0.5cm, is measured using either M-mode or 2D imaging. Acute MI involving RV may cause RV dilatation and RV thinning. RV free wall thickness can be assessed from t apical 4 chamber view, parasternal long-axis view or the subcostal view(preferred).

(2) Right Ventricle Dimensions

RV dimension assessment is very difficult for the following reasons. Firstly, the complex anatomical shape – crescentic shape of the cavity. Secondly, the presence of irregular endocardial surface. Thirdly, the complexity in its contraction mechanics comparable to that of bellows. Lastly its location immediately behind the sternum also poses a problem in the evaluation of RV anatomy and function.

RV is crescentic in its minor axis, but along its long axis it is complex.No simple geometric three dimensional figure accurately represents this chamber. Contraction is also complex. Relatively small movements produce large ejection volumes. Normally the RV is two thirds of the size of Left ventricle. To measure the RV dimensions and volumes, both the area – length and Simpson's rule has been employed..

Qualitative assessment of RV size is easily accomplished from the apical 4-chamber view. In this view, RV area or midcavity diameter should be smaller than that of the LV. In cases of moderate enlargement, the RV cavity area is similar to that of the LV and it may share the apex of the heart. As RV dilation progresses, the cavity area will exceed that of the LV and the RV will be apex forming. Quantitative assessment of RV size is also best performed in the apical 4-chamber view. Care must be taken to obtain a true non foreshortened apical-4 chamber view, oriented to obtain the maximum RV dimension, before making these measurements. Measurement of the midcavity and basal RV diameter in the apical 4-chamber view at end diastole is a simple method to quantify RV size. In addition, RV longitudinal diameter can be measured from this view.

Table shown below provides normal RV dimensions from the apical 4-chamber view.

RV Dimensions (cm)	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
Basal RV diameter	2.0-2.8	2.9-3.3	3.4-3.8	≥3.9
Mid RV diameter	2.7-3.3	3.4-3.7	3.8-4.1	≥4.2
Base to apex length	7.1-7.9	8.0-8.5	8.6-9.1	≥9.2

Normal RV areas and fractional area change are shown in Table shown below.

Variable	Reference Range	Mildly Abnormal	Moderately Abnormal	Severely Abnormal
RV diastolic area, cm^2	11-28	29-32	33-37	≥38
RV systolic area, cm^2	7.5-16	17-19	20-22	≥23
RV fractional area change, %	32-60	25-31	18-24	≥17

RV dimensions and % F S of different axis of the RV cavity were assessed from the apical four- chamber view according to the method proposed by **Bommer *et al***. In this view, the RV internal borders were

carefully underlined after optimal gain setting in technically adequate images. The long axis dimension was measured from the apex of the RV to the midpoint of the tricuspid valve annulus plane. The maximal short axis dimension was measured between the interventricular septum and the free lateral wall at the mid point of the long-axis dimension. RV axis diameters were measured at end-diastole (at the peak of R wave) and at end-systole (at the point of maximal inward motion of the endocardium). The % F S for each axis was calculated according to the following formula:

$$\% FS = \frac{RV\ EDA - RV\ ESA}{RV\ EDA} \times 100$$

RV fractional area change measured in the apical 4-chamber view is a simple method for assessment of RV function that has correlated with RV EF measured by MRI and has been related to outcome in a number of disease states. RV size may be assessed by TEE in the mid esophageal 4-chamber view also.

(3)RV wall motion

To assess regional wall motion the RV was divided into four segments: diaphragmatic, free lateral, apical. and interventricular septum walls. RV regional wall motion analysis (qualitative) was characterized as normal (>40% thickening with systole); hypokinetic (10-30%

thickening), akinetic (<10% thickening), dyskinetic (paradoxic expansion in systole) or aneurysmal . RV involvement was diagnosed when at least one new regional wall motion abnormality was observed, except the interventricular septum alone. When a patient presents with a STEMI ,the affected myocardium is usually akinetic or dyskinetic. The absence of wall motion abnormalities during chest pain usually but not always excludes MI and the presence of regional wall motion abnormalities has a high sensitivity for detecting MI although it is not specific. In the vast majority of patients with RVI, the wall motion abnormalities that initially manifest on echocardiography reverse within 3 months.

Indices of tricuspid annular motion including TAPSE, DTI, and MPI are useful for the assessment of RV function.

(4) <u>Tricuspid Annular Peak Systolic Excursion</u>

Qualitative assessment of RV systolic function can be made by estimating the displacement of the tricuspid annulus. Tricuspid annular motion refers to the distance ,the tricuspid annulus moves in the antero-posterior direction. In systole, the tricuspid annulus will normally descend toward the apex 1.5 to 2.0 cm. Tricuspid annular excursion of less than 1.5 cm has been associated with poor prognosis in a variety of cardiovascular diseases. Since the tricuspid valve moves

toward the RV apex during ventricular systole as lengthwise shortening of both the interventricular septum and RV free wall, it is intuitively evident that TAPSE or TAPSE per time must be related to RV EF[42] .The level of excursion of the tricuspid valvular plane during systole (TAPSE, in mm) corresponds with RV ejection fraction (5 mm ~20% RV ejection fraction, 10 mm ~30% RV ejection fraction, 15 mm ~40% RV ejection fraction, and 20 mm ~50% RV ejection fraction)[43][44].

(5)**Tricuspid annular fractional shortening**

Tricuspid fractional shortening is an assessment of the difference between the maximal and minimal distance between the tricuspid annuli during the cardiac cycle.

(6)Trans tricuspid inflow velocities

Because the effective orifice area of the tricuspid valve is substantially greater than that of the mitral valve, the inflow velocities are lower than for the mitral valve. As for the mitral valve, however, the normal pattern consists of relatively higher early inflow (E-wave) and a lower velocity flow concordant with atrial systole (A-wave). Evaluation of right ventricular diastolic function is conventionally based on the Doppler transtricuspid flow velocity profile. Variables measured include peak velocity of early filling (E velocity),peak velocity of late filling due to atrial contraction (A velocity), E/A ratio and deceleration time of early

filling (Edt). The velocities across the tricuspid valve are significantly lower than across the mitral valve and tricuspid Edt is longer than mitral Edt. The tricuspid flow parameters do not appear to be affected by age but respiration causes pronounced variability and measurement should be made at end expiratory apnoea. The normal filling pattern is affected by pre-load, afterload and compliance of the ventricular myocardium.

(7)MYOCARDIAL PERFORMANCE INDEX – TEI INDEX.

Studies have found several novel echocardiographic and Doppler measurements of RV function to be risk factors for heart failure, independently of traditional risk factors. **Tei *et al*[(45)]** studied, in 1995, the ability of a new Doppler index of combined systolic and diastolic function (Tei index) to separate patients with normal ventricular function from patients with heart failure. They showed that the separation between normal individuals and patients with dilated cardiomyopathy, by use of the Tei index, was superior to other available indexes. The Tei index[(46)] has the advantages of being less affected by age, heart rate, and preload than conventional Doppler measurements, and being calculated from 2 well-defined time intervals, and the index has an excellent reproducibility(interobserver variability<5%) **[(47)].** MPI has been shown to have prognostic value in patients with coronary heart disease, dilated cardiomyopathy, amyloidosis, coronary heart disease and symptomatic

heart failure as well as in the general population. MPI provides prognostic information independently of other measurements of cardiac function and of traditional risk factors for heart failure. Therefore, MPI seem to be a clinically relevant measurement of global ventricular function and may prove to be a valuable tool in assessing the risk of developing right heart failure.

Our knowledge of the natural history of heart failure indicates that both asymptomatic systolic and diastolic dysfunction can precede the onset of overt heart failure. The utility of MPI is comparable to simultaneous cardiac catheterization measurements of RV function as the MPI was found to reflect both systolic and diastolic function.

MPI mirrors both the depolarisation and repolarisation process. It seems like changes in cellular Ca^{2+} handling in the myocardium underlie much of the abnormal contractility and relaxation. In the failing heart, the contraction and relaxation becomes slower explaining why MPI increases with deterioration of cardiac function. There is evidence that sub-clinical depolarisation (and repolarisation) defects to be early phenomena in the natural history of heart failure as noted above.

MPI is a unit less number reflecting the global performance of the ventricle. It was devised in the mid 1990's **(Tei *et al*)**. It is a simple

index which incorporates both systolic and diastolic parameters and can be applied to either LV or RV. Several studies have used this index as a prognostic indicator of LV performance.The utility of MPI as an indicator of global RV performance is an area of interest in the recent past.

The ratio of isovolumic contraction time (IVCT) and ejection time (ET) was closely correlated to +d*P*/d*t* (reflecting systolic function) and the ratio of isovolumic relaxation time (IVRT) and ejection time was closely correlated to –d*P*/d*t* and *s* (reflecting diastolic function). Therefore, MPI may be considered the sum of an index reflecting systolic function and an index reflecting diastolic function. Thus, the superior predictive capacity of MPI could be explained by the fact that MPI reflects global function, while other measurements are limited to reflect mainly either LV systolic or diastolic function.

$$MPI = \frac{IVRT + IVCT}{ET}$$

According to the above equation, systolic dysfunction is characterised by the prolongation of IVCT and decrease in ET. Whereas the diastolic dysfunction is characterised by the lengthening of IVRT. Presence of both is indicated by an increase in MPI. The normal value of

MPI of Left ventricle is 0.39 +/- 0.08. For the right heart, the normal values are 0.28 ± 0.04. **Özdemir *et al*(2003)[48]** ;

Fan Ying *et al* 2005[49], have demonstrated in their study that an MPI of > 0.70 may diagnose RVMI and proximal right coronary artery disease with high sensitivity and specificity.

Thus, MPI is a reliable and easily assessable measurement of global ventricular function, and as such, suitable for large-scale examinations. Nevertheless, further studies are needed in order to define the role of MPI in clinical practice. Clinically significant age- and gender-specific cut-off points need to be defined ; furthermore it has to be determined if pharmacological and/or non-pharmacological interventions lower MPI and whether lowering MPI modifies the risk associated with a high MPI. The potential problems with Tei index is that it is invalidated by heart block and arrhythmias. Primary valvular diseases also leads to difficulties in its interpretation.

IVCT and IVRT

Isovolumetric contraction phase is the earliest phase of ventricular systole in which the ventricle contracts as a closed chamber without any change in the volume of the chamber. The isovolumic contraction time corresponds to when calcium enters the myoplasm from the sarcolemma.

Traditionally IVRT is recorded as the aortic valve closure and mitral valve opening time in the Left ventricle. M Mode echocardiography and pulsed wave Doppler were used for this purpose. Normal values approximates 65 $\pm$ 20 msec. Isovolumic relaxation time reflects the removal of Ca^{2+} from the myoplasm by Ca^{2+}-ATPases. The IVCT increases and the RV ejection time decreases in systolic dysfunction. Most abnormalities of diastolic function are manifested in an abnormally slow rate of pressure decline. These changes are reflected in a prolongation of the IVRT. As global myocardial dysfunction progresses, the value for the index of myocardial performance increases, due to changes in all three time interval components used for its calculation.

Recently continuous wave Doppler echocardiography has been used to measure IVRT. The apical 5 chamber view is used for this purpose. Measurements made by this method is comparable to that made by invasive methods. IVRT represents the earliest phase of ventricular diastole. Abnormalities of this index has been described as a non invasive predictor of diastolic dysfunction. However, measurement of IVRT as the sole indicator of diastolic dysfunction is limited, since no information on ventricle filling is provided.

(8)Tricuspid Lateral Annular and Septal Annular Velocity

Contraction of both the septum and the RV free wall contributes to the RV ejection fraction. Myocardial velocity determined by Doppler tissue imaging (DTI) is a new technique that has been used recently to analyse left ventricular function. The development of DTI opens up the possibility of also assessing RV function. The myocardial velocities obtained by pulsed-wave TDI from the RV free wall at the level of the tricuspid annulus in the apical four-chamber view show the longitudinal dynamics of the right ventricle, which is not seen with visual scoring. In studies assessing RV function by using techniques other than TDI, the movement of the tricuspid annulus has been shown to represent the global RV function. It has been shown that tricuspid annular systolic velocity (Sm),early and late diastolic velocities (Em and Lm) decreases in patients with inferior MIs compared to those with anterior MIs, and in those with RVMIs compared to those without RVMIs.

Similar decrease has been seen in the interventricular septal annulus systolic and early diastolic velocity. Many studies **(Alam *et al*, Ozdemir *et al*)**[1] [48] have shown that an Sm of < 12 cm/s showed RVMIs with high sensitivity (81%) and specificity (82%); In addition, the negative predictive value was also high (92%).

A decrease in the systolic velocity at the tricuspid annulus not only allows for diagnosis of RVI but also suggests worse mortality outcome

(Hisham,2005). A decrease in the early diastolic velocities (Em) of the RV free wall shows the development of RV diastolic dysfunction in those with RVMIs and in those who experienced MIs due to a proximal right coronary artery lesion.

Other Findings to be noted

- Paradoxical motion of the interventricular septum may be seen after a RVMI.
- Tricuspid regurgitation.
- Pericardial effusion.

Transient pericardial effusion is not uncommon after acute MI. It is seen typically in transmural or Q-wave MI and only rarely in non Q-wave MI. Careful surveillance studies have demonstrated that 30% to 40% of patients with acute transmural infarction will have transient accumulation of small amounts of pericardial fluid. The genesis of this effusion is assumed to be epicardial inflammation, and it may be seen in the absence of any symptoms specific for acute pericardial disease.

Other Investigations in Myocardial Infarction

Several **radionuclide imaging** techniques (Myocardial perfusion imaging with ^{201}Tl or ^{99m}Tc-sestamibi, Radionuclide ventriculography, carried out with ^{99m}Tc-labeled red blood cells) are available for evaluating patients with suspected STEMI. However, these

imaging modalities are used less often than echocardiography because they are more cumbersome and lack sensitivity and specificity in many clinical circumstances.

Myocardial infarction can be detected accurately with **high-resolution cardiac magnetic resonance imaging** using a technique referred to as late enhancement. A standard imaging agent (gadolinium) is administered and images are obtained after a 10-min delay.Since little gadolinium enters normal myocardium where there are tightly packed myocytes,but does percolate into the expanded intercellular region of the infarct zone, there is a bright signal in areas of infarction that appears in stark contrast to the dark areas of normal myocardium.

Eric Larose *et al* 2007 [(50)] in their study showed that RV function assessed late after clinical MI is an important predictor of post-MI mortality, independent of patient age, LV infarct size, and LVEF. Evaluation of RV function using CMR may improve the risk stratification of patients with MI beyond current practice and refine their medical management. Although echocardiographic assessment of tricuspid annular excursion and radionuclide technique can both assess the RV in this setting, cardiac magnetic resonance imaging (CMR) can provide an improved quantitative and volumetric method of assessing RV size and function.

AIMS AND OBJECTIVES

(1) To study the clinical profile of patients with IWMI.

(2) To evaluate the function of Right Ventricle in all IWMI patients.

MATERIALS AND METHODS

About 35 patients who got admitted with IWMI to the Dept. of Medicine and Dept. of Cardiology between February to July 2008 and 10 normal subjects were chosen and studied.

The type of study : Cross sectional study

Selection criteria and study population

Inclusion criteria

Thirty five patients (mean age 54.2 $\pm$ 12) who were admitted to our hospital and found to have IWMI and ten healthy individuals were subjected to a complete echocardiographic examination. IWMI was diagnosed by the presence of ST-segment elevation greater than 1 mm in 2 anatomically contiguous leads or the presence of new Q waves in leads II,III and AVF. ST-segment elevation >1mm in lead V_4R was used as the main criteria for the diagnosis of RVMI . In our study group, RV involvement in varying proportions were found in many patients with IWMI as determined by echocardiography when compared with the healthy subjects.

Exclusion criteria

Patients with anterior/anteroseptal/lateral wall MI, chronic obstructive pulmonary disease, pulmonary hypertension, cardiomyopathy, valvular heart disease or congenital heart disease with left to right shunt lesions were all excluded.

The Institutional Ethical Committee approved the study and all patients gave informed consent to undergo evaluation.

Methods :

All the patients included in the study were subjected to detailed history taking , complete physical examination, investigations like complete blood count, RBS, RFTs, fasting total cholesterol, cardiac markers like total creatine kinase and CK-MB. ECG was used to determine whether these patients are having RVMI/PWMI or both.

All patients underwent a complete transthoracic echocardiographic study including two-dimensional, M Mode, pulsed wave doppler and DTI were done using ALOKA SSD 4000 echocardiography machine. Continuous ECG monitoring of the patients was done during the procedure with the patient lying in standard left lateral decubitus

procedure. Standard two-dimensional echocardiographic evaluation of (RV) size and function was performed. Right ventricle was visualised by the apical four chamber view.Modified Simpson's rule was employed for estimating RV area . In addition, right ventricular end-diastolic (RV EDA) and end-systolic areas (RV ESA) were measured from the apical 4-chamber view to calculate right ventricular fractional area change (RV FAC). M mode and two dimensional echocardiography were used to document presence or absence of pericardial effusion. The free fluid in the pericardial cavity was visualized as an echo-free space using the parasternal long axis and short axis views. Paradoxical septal motion was visualized in the parasternal long axis view .

The tricuspid annular motion was recorded at the RV free wall from the apical 4-chamber view. In the real-time 2-dimensional apical 4-chamber view, the M-mode cursor was placed through the tricuspid annulus in such a way that the annulus moved along the M-mode cursor. The displacement of the tricuspid annulus was recorded in M-mode. The total displacement was measured by using the leading edge of the echoes.

Tricuspid Annular Fractional Shortening was calculated by taking the difference between tricuspid annular diameter measured during diastole and systole.

The presence or absence of RV wall motion abnormality was assessed qualitatively from the parasternal long-axis view, parasternal short-axis view at the level of mid-ventricle (including the anterior and posterior sites near the interventricular septum excluding the free wall because of difficulties in visualisation), and apical 4-chamber view for the free wall. The wall motion was judged to be normal, hypokinetic,akinetic, dyskinetic or aneurysmal at any of the RV sites.

With the use of the apical 4-chamber view, pulsed-wave Doppler trans tricuspid flow velocities were recorded by placing the sample volume between the leaflet tips in the center of the flow stream. The trans tricuspid peak rapid filling velocity (E), peak atrial filling velocity (A), E-wave deceleration time, and E/A ratio were recorded.

From the apical 4-chamber view, the DTI cursor was placed at the tricuspid annulus of the RV free wall in such a way that the annulus moved along the sample volume line. During systole, a major positive velocity (Systolic 'Sm') was recorded when the annulus moved toward the cardiac apex. During diastole, when the annulus moved toward the base away from the apex, 2 major negative velocities were recorded: one during the early phase of diastole [Early diastolic (Em)] and another during the late phase of diastole [Late diastolic (Am)].Similar

measurements was recorded from the septum by placing the DTI cursor at the septal site of the annulus.

Isovolumetric contraction time (IVCT), isovolumetric relaxation time (IVRT) and ejection time (ET) were measured by keeping the DTI cursor at the tricuspid annulus and subsequently RV Myocardial Performance Index (MPI) or the Tei index was calculated using the standard formula MPI = IVRT+IVCT / ET. The reference limits used for the MPI of right ventricle is 0.28 ± 0.04.

The reference limits for various echocardiographic indices and parameters used in this study were adopted from the standard echocardiographic manuals.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2002).Using this software, frequencies, percentage, mean, standard deviation, $\mathbf{x^2}$ and **'p'** values were calculated. A **'p'** value less than 0.05 is taken to denote significant relationship.

RESULTS

The results of clinical evaluation of the 35 patients and 10 healthy individuals are shown below.

GROUP A : INFERIOR WALL MI PATIENTS

GROUP B : NORMAL HEALTHY INDIVIDUALS

A : CHARACTERISTICS OF STUDY POPULATION –

INFERIOR WALL MI PATIENTS = GROUP A

The **table 1** and **fig 1** shows the age wise distribution of IWMI patients in our study. The mean of the age of patients in our study is 54.2 $\pm$ 12 years.

TABLE 1

AGE DISTRIBUTION

Age	Study cases	
	No.	**%**
Upto 30 years	2	5.7
31-40	3	8.6
41-50	6	17.1
51-60	16	**45.7**
Above 60 years	8	22.9
Total	35	100
Mean	**54.2 years**	
S.D.	**12 years**	

The **table 2** and **figure 2** shows the sex distribution of the IWMI patients. Males constituted the majority (80%).

Table 2: Sex Distribution

Sex	Study cases	
	No:	**%**
Male	28	**80**
Female	7	20
Total	35	**100**

Table 3 shows the symptom analysis of IWMI patients. Many patients had more than one symptom at the time of presentation with chest pain being the most common**.**

Table 3 : Symptoms

Symptoms	Study cases	
	No:	%
Chest pain	27	**77.1**
Dyspnea	11	19.9
Giddiness	9	25.7
Fatigue	1	2.9
Epigastric pain	-	2.9
Diaphoresis	21	60
Syncope	-	-
Vomiting	5	14.3
Palpitation	8	22.9
Pedal Edema	1	2.9

Table 4 : KILLIP's Class at the time of admission

Class	Study cases	
	No:	**%**
I	27	**77.1**
II	5	14.3
III	-	-
IV	3	8.6
Total	35	100

Most of the patients(77.1%) admitted with IWMI were in Killip's Class I.(**table 4**;**fig 3**)

Among the 35 patients, 11 patients (31.4%) were hypertensives and 8 patients (22.9%)were diabetics. Of the 35 patients,19 were smokers and 12 of them were alcoholics. Among the 7 female patients,4 were post-menopausal. Complete blood count (CBC) showed anemia in 17 patients (48%) and CBC was normal in the rest.The renal function testing revealed no abnormalities in the study population. Random blood glucose estimation revealed that 10 patients (28.57%) were having plasma glucose above 200 mg/dl.

Table 5 shows the fasting total cholesterol levels in the IWMI patients.29 patients(82.85%) were having their cholesterol in the desirable range(<200 mg/dl) ; 5 had borderline high values(200-239); 1 had high (≥240) cholesterol levels.

Table 5 : Fasting total cholesterol levels in IWMI patients

Fasting total Cholesterol (mg%)	Study cases	
	No:	**%**
<200	29	**82.85**
200-239	5	14.29
≥240	1	2.86
Total	35	**100**

Cardiac markers estimation

Creatine kinase –total revealed high values (ie >294 U/L)in 9 male patients and high values (>238U/L)in 3 female patients. Creatine Kinase MB fraction were higher(>12 IU/L) in 31 patients.

ELECTROCARDIOGRAPHY : ECG revealed changes of RVMI in 32 patients (91.42%) ; that of PWMI in 14 patients (40 %).

ECHOCARDIOGRAPHY

Comparison of various echocardiographic parameters between Group A (inferior wall MI patients) and Group B (normal healthy individuals)

Table 6 : Comparison of TAM and TAFS between Groups A and B

Parameter	Group A			Group B			'P'
	Range	Mean	S.D.	Range	Mean	S.D.	
TAM (mm)	2-24	16.3	3.5	22-23	23.4	1.1	**0.0001 Significant**
TAFS	4 - 57.14	25.8	11.2	14.4 – 16.2	15.4	0.75	**0.0017 Significant**

TAM was significantly decreased in Group A subjects(P value 0.0001)compared to that of Group B subjects**.(Table6)(Fig 4).**TAFS measurement showed statistically significant change (P value 0.0017) between the two study groups**.(Table 6)(Fig 4).**

The difference in RV dimensions between group A and B were statistically significant in case of RV base (P value 0.0001), RV EDA (P value 0.0008), RVFAC (P value 0.0049, RV free wall thickness (P value 0.0001) and not significant in case of RV long axis (P value 0.2284) and RV ESA (P Value0.8057) **(Table 7) (Fig 5).**

Table 7 : RV DIMENSIONS

Parameter	Group A			Group B			'P'
	Range	Mean	S.D.	Range	Mean	S.D.	
RV base	2.4 - 4.4	3.29	0.49	2.2 – 2.8	2.48	0.22	**0.0001** **Significant**
RVLA	5.2 – 8.9	7.18	0.82	7.4 – 7.8	7.57	0.18	**0.2284** **Not significant**
RV EDA	10.4 – 23.7	14.4	2.7	15 - 18	17.1	1.1	**0.0008** **Significant**
RV ESA	4.82 – 14.01	8.9	2.25	8 -10	8.9	0.74	**0.8057** **Not significant**
RV FAC	19.3 – 57.9	38.1	10.7	46.7 - 50	48	2.33	**0.0049** **Significant**
RV WT	3 - 7	4.37	0.84	0.3 – 0.4	0.38	0.04	**0.0001** **Significant**

Abnormalities in wall motion was seen in 29 patients (82.8%) in group A; while all the subjects in group B showed normal wall motion of the RV.The difference in wall motion between the two groups was statistically significant (P value 0.0001) **[Table 8]**. Paradoxical motion of the interventricular septum was not seen in both groups .

Table 8 : RV Wall Motion

Wall Motion	Group A		Group B	
	No.	**%**	**No.**	**%**
Hypokinetic	25	71.4	-	-
Akinetic	4	11.4	-	-
Normal	6	17.1	10	**100**
Total	35	100	10	**100**
'p'	**0.0001 (Significant)**			

Pulsed wave Doppler estimation of transtricuspid peak rapid filling velocity (E) , peak atrial filling velocity (A), E wave deceleration time and E/A ratio ,all showed statistically significant change between the two study groups**.[Table 9]**.

Table 9

Parameter	Group A			Group B			'P'
	Range	**Mean**	**S.D.**	**Range**	**Mean**	**S.D.**	
E	0.37 – 0.74	0.54	0.09	0.46 –0.52	0.49	0.02	**0.0051 Significant**
A	0.24 – 0.68	0.42	0.07	0.33– 0.39	0.36	0.02	**0.0327 Significant**
E/A	0.61 – 2.28	1.35	0.35	1.1– 1.23	1.14	0.04	**0.0426 Significant**
E wave DT	185 - 325	250.5	39.2	160 - 192	171	9.4	**0.0001 Significant**

Statistically significant (P value 0.0001) increase in MPI values was seen in Group A (mean 0.6 $\pm$ 0.25) compared to that of Group B (mean 0.22$\pm$0.02) **.[Table 10].**

Table 10

Parameter	Group A			Group B			'P'
	Range	Mean	S.D	Range	Mean	S.D	
IVCT	18 - 85	53.1	20.9	40 - 44	42.1	1.4	**0.1126 Not significant**
IVRT	32 - 260	87	48.1	32-38	34.7	1.8	**0.0001 Significant**
ET	128 - 349	234.5	44.8	320 - 362	343.8	15	**0.0001 Significant**
MPI	0.14 – 1.26	0.6	0.25	0.19 – 0.25	0.22	0.02	**0.0001 Significant**

Estimation of the tricuspid annular velocity showed statistically significant reduction in peak systolic velocity(Sm) [P value 0.0002],early diastolic velocity(Em) [P value 0.0006] and late diastolic velocity(Am) [P value 0.0059]. **[Table 11] (Fig 6).**

Table 11 : Tricuspid Lateral Annular velocity

TAV	Group A			Group B			'P'
	Range	Mean	S.D.	Range	Mean	S.D.	
TAV Sm	2 -16	11.5	2.3	13.8 – 14.6	14.2	0.3	**0.0002 Significant**
TAV Em	4 - 22	10.4	3.6	13 – 14.2	13.6	0.4	**0.0006 Significant**
TAV Am	6 - 41	12.3	6.4	13.8 – 14.4	14.1	0.2	**0.0059 Significant**

Estimation of the tricuspid septal annular velocity showed statistically significant reduction in peak systolic velocity(Sm) [P value 0.0004],early diastolic velocity (Em),[P value 0.0003] and late diastolic velocity(Am) [P value 0.0001]. **(Fig 7)**. **[Table 12].**

Table 12 : Tricuspid Septal Annular Velocity

Septal Annular Velocity	**Group A**			**Group B**			**'P'**
	Range	**Mean**	**S.D.**	**Range**	**Mean**	**S.D.**	
IVS Sm	5 - 11	7.5	1.7	8.8 - 10	9.6	0.5	**0.0004 Significant**
IVS Em	4 - 13	7.5	2.4	11 – 11.4	11.1	0.2	**0.0003 Significant**
IVS Am	4 - 13	8.3	1.9	11.2 - 12	11.8	0.3	**0.0001 Significant**

DISCUSSION

Demographics

The demographics of ACS mainly reflect the risk factors associated with atherosclerosis. Most patients hospitalized are between 50 and 70 years-old, with patients having unstable angina being slightly older. Because the Global Use of Strategies to Open Occluded Coronary Arteries **(GUSTO) IIb study,1996**[51] simultaneously enrolled patients across the spectrum of ACS, its data are particularly relevant to assess the underlying demographics. Of the 12,142 patients enrolled in this study, the median ages for those with STEMI and no ST segment elevation (patients with unstable angina or NSTEMI) were 63 and 66 years, respectively (Figure 5). Perhaps because females represent a greater percentage of the elderly population, they have a relatively greater presence in the ACS patients without STEMI. While men more commonly have ACS than women, the ratio of men to women with no ST-segment elevation (2:1) is lower than for patients with STEMI (3:1).

According to World Health Report 2002, cardiovascular diseases (CVDs) will be the largest cause of death and disability by 2020 in India. In 2020 AD, 2.6 million Indians are predicted to die due to coronary heart disease which constitutes 54.1 % of all CVD deaths. Nearly half of these deaths are likely to occur in young and

middle aged individuals (30-69 years). Currently Indians experience CVD deaths at least a decade earlier than their counterparts in countries with established market economies[72].Although no major differences in total prevalence was reported, age specific rates show a decline in males less than 40 years old. There was an increase in CHD prevalence in those aged 40–59 years among men.

Our study also shows an increased prevalence of MI in patients aged > 40 years (85%).Patients aged 40-59 yrs constitutes 63 % of the total patients similar to the western and national picture .

80% of our patients are males(.M:F=4:1) compared to the 3:1 ratio observed in the **(GUSTO) IIb study**[51]. Of the 7 female patients,5 are post menopausal and among the other 2 patients ,one is a known diabetic.

The most common symptom of any patient presenting with MI is central chest pain lasting >20 minutes.77 % of our patients are having their presenting symptom as chest pain.Other common presenting symptoms are dyspnea(19.9 %),giddiness(25.7%) and palpitation(22.9 %).

Zaacks *et al* 1999 [52] observed in their study that classic angina history is obtained in only about half of the patient presenting with ACS. The other half of the patients present with atypical presentation and picking up these patients, diagnosing and treating them appropriately is

challenging.I5-30% of patients may not present with typical symptoms in case of MI[53].

CC Escosteguy *et al*.2003[54] in his sectional study of a sample with 391 randomly drawn medical records of the hospitalisations due to acute myocardial infarction recorded in the hospital information system in 1997 found that 82.6% presented in the Killip's class I stage. Majority of patients (77.1%) in our study are in Killip's class 1 stage at the time of presentation similar to the picture in the above mentioned study.

In the **Gusto II b trial**[51],CAD risk factors—hypertension, hypercholesterolemia, and tobacco use—are present in 30–40% of patients with an ACS, whereas diabetes was present in 15–20%. In our study, 31% of patients were known hypertensives,22% were known diabetics,34% were chronic smokers and 17 % patients were having the cholesterol levels above the desirable range showing similar picture like that of the Gusto II b trial.

Echocardiography

Echocardiography developed over the last three decades has become an extension of the physical examination in the modern evaluation of a patient with heart disease. The review of the various studies shows that this unique non invasive technique is useful in knowing the anatomical and pathophysiological alterations and in the hemodynamic evaluation of any disease leading to heart failure. Although RV infarction is not an uncommon condition, hemodynamically significant RV infarction is an infrequent consequence of inferior MI. Occlusion of the proximal right coronary artery might produce inferoposterior left ventricular infarction in association with RV infarction and depressed RV function. Assessment of RV function by echocardiography is complicated because of the complex geometry. Contraction of both the septum and the RV free wall contributes to the RV ejection fraction.

Motion of the tricuspid annulus toward the cardiac apex in systole is an expression of RV contraction along the long axis. Previously, RV function has been evaluated by use of the amplitude of the tricuspid annular motion determined by M-mode echocardiography. The advantage of recording the tricuspid annular motion is that it is devoid of myocardial dropout.

In our study, significant reduction in TAM is seen in IWMI patients compared to healthy individuals. This is probably an expression of decreased RV function along the long axis in inferior MI. This is similar to the TAM change observed by **Mahbubul Alam *et al* 2000.** [(1)] in patients with IWMI.

Kaul *et al* [(55)], using cross sectional echocardiography and radionuclide angiography, observed that the measurement of tricuspid annular motion could reflect systolic function.

Ghio *et al*,2000[(56)], using M mode cross sectional echocardiographic and thermodilution derived right ventricular ejection fraction measurements, demonstrated that tricuspid plane motion can be considered a physiological index of right ventricular function.

Samad BA *et al*, 2002[(57)]in his study on patients with acute MI showed that recordings of TAM by echocardiography can be used to assess RV function in most patients and have a predictive value after acute MI.

More recently**, Meluzin *et al*,2001**[(58)]and **Moustapha *et al*,2001**[(59)] obtained similar results .The clinical importance of assessing annular excursion is also documented by **Willenheimer *et al*,1997**[(60)] and **Karatasakis *et al*,1998**[(61)] .

Smith JL *et al* ,2003[44] has found that measurement of tricuspid annulus motion is an easy way to estimate right ventricular ejection fraction .

Sripal Bangalore *et al.*2007,[62] has observed that in the presence of an abnormal left ventricle patients with an abnormal RV had a worse prognosis than those with a normal RV; Abnormal RV was a significant predictor of events independent of LV ischemia and ejection fraction .

RV dilatation was observed in all patients with RVMI in a study conducted by **Jugdutt BI *et al,***[63] .Our patients has also shown a significant increase in RV base size compared to the control subjects.

RVFAC is significantly decreased in Group A compared to that of Group B. **Leonardo A.M *et al,* 2002**[12] observed that in many patients after a myocardial infarction (more in patients with IWMI) , many patients showed a significant decrease in RV fractional area change. They observed that RV function remained an independent predictor of total mortality, cardiovascular mortality and heart failure. Each 5% decrease in the RV FAC was associated with a 16% increased odds of cardiovascular mortality (95% confidence interval 4.3% to 29.2%; P = 0.006) .

In our study ,29 out of the 35 patients (82.8 %) have either hypokinesia or akinesia of the RV showing involvement of RV in the infarction. **A Arditti *et al.* 1985,**[64] has shown in their study that 58 percent of patients with acute inferoposterior MI had 2D echo regional wall motion abnormalities compatible with RV dysfunction.

RV wall thickness measured in our study showed normal values (<5 mm)[40].

Measurements of trans tricuspid inflow velocities in our patients shows significant diastolic dysfunction of the right ventricle.

The Doppler index of myocardial performance (Tei index or myocardial performance index) is yet another parameter that can be used for evaluation of RV performance[40][42][65].

In our study, the mean RV MPI is 0.60 ± 0.25 compared to the normal value of 0.28 ± 0.04 showing global RV dysfunction in majority of patients.

Chockalingam, A.B *et al.* 2004,[32] showed significant increase in RV MPI in patients with RVMI. RV MPI $\geq$ 0.30 has high sensitivity (82%) and specificity (95%) for the diagnosis of RVMI in the presence of acute IWMI .

Shiro Yoshifuku *et al*.2003,[66] showed in their study that RV Tei index was significantly increased in patients with RV infarction compared with those without (0.53 ± 0.15 vs 0.38 ± 0.14, p <0.05).

Ozdemir *et al* 2003[48] and **Fan Ying *et al*. 2005** [49] showed the usefulness both of peak myocardial systolic velocity (Sm) and of the myocardial performance index (MPI) of the right ventricle measured by pulsed-wave tissue Doppler imaging (TDI) in assessing right ventricular function ; The sensitivity, specificity, negative predictive value, and positive predictive value of an MPI of > 0.70 in the diagnosis of RVMI were calculated as 94%, 80%, 97%, and 63%, respectively and in the diagnosis of the proximal right coronary artery as the infarct related artery, those values were 78%, 91%, 83%, and 88% respectively; A Sm<12 cm/s and an MPI > 0.70 obtained by TDI may define RVMI concomitant with acute inferior MI, and the infarct related artery.

Recently, myocardial velocity recorded by either color-coded or pulsed-wave DTI has been used to assess left ventricular function. However, the effect of MI on RV velocities estimated by DTI is not known.

Tricuspid annular velocity is a surrogate for global right ventricular systolic function and has been found to be lower in patients with inferior myocardial infarction, especially if there is evidence of right ventricular involvement[50]. A good correlation between annular velocity and radionuclide ejection fraction also has been reported [43].

In our patients with IWMI, significant reduction is seen in peak systolic, early diastolic and late diastolic tricuspid annular velocity on comparison with healthy subjects. Similar change was also seen in patients with IWMI by **Mahbubul Alam *et al.*2000[1].**

Meluzin J *et al.*2001 [58], observed that the peak systolic tricuspid annular velocity significantly correlates with the right ventricular ejection fraction assessed by first-pass radionuclide ventriculography, and its value <11·5 cm / s enables right ventricular dysfunction to be predicted (ejection fraction <45%) with a good sensitivity of 90% and a specificity of 85% in patients with heart failure.

Hisham Dokainish *et al.*2005 [31] observed that decreased RV systolic annular velocity on TDI detects RVMI in first left ventricular acute inferior MI and predicts cardiac death or rehospitalisation at 1 year.

The interventricular septum was traditionally been considered to be a part of the left ventricle. However, contraction of the septum also contributes to the RV ejection[23].In the current study, a

significant decrease in peak systolic / early and late diastolic velocity of the septum is seen in inferior MI patients.

Mahbubul Alam *et al*. 2000[1] in his study on patients with IWMI also observed a similar reduction in the peak systolic and early diastolic velocities of the septum compared to healthy individuals.

Limitations of the study

Several important limitations of these studies should be acknowledged.

1. The number of patients were relatively small in our study
2. ECG criteria was used for selecting IWMI patients, no coronary angiography was performed during the acute phase of the infarction for analysis of the state of right coronary artery.
3. The patients with IWMI were compared with healthy individuals ; IWMI patients were not evaluated separately based on the presence or absence of RVMI.
4. The use of fractional area change as an index of global right ventricular systolic function has a limitation of being highly afterload dependent.
5. Echocardiographic estimation was done 2-4 days after the infarction; It is possible that some of the patients had already recovered from the damage in the right ventricle, because RV recovery generally occurs quickly after MI.

CONCLUSION

We conclude that many of our patients with IWMI have significant impairment of RV function. RV involvement in a patient with IWMI has been reported to be an independent predictor of major complications and in-hospital mortality after acute inferior MI. RVMI is associated with increased risk of death, shock, ventricular tachycardia or fibrillation and atrioventricular block. Echocardiography helps one to identify such patients easily and to treat them immediately. As discussed such patients have a decreased tricuspid annular motion, tricuspid annular velocity and RV MPI compared with normal healthy individuals. This probably reflects a reduced RV function after RV infarction. So we conclude that an immediate echocardiography can help us to identify and treat such patients with RV involvement and thereby to reduce the morbidity and mortality.

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APPENDIX I – PROFORMA

EVALUATION OF RV FUNCTION IN IWMI PATIENTS

Name: Age: Sex:
Occupation:

Address: IP/OP No: CD.No:

Complaints (NYHA functional class)

Chest pain / Dyspnoea / Giddiness / Fatigue / Epigastric pain /

Diaphoresis Syncope / Vomiting / Palpitation / Pedal edema

Past History

SHT / DM /CAD/RH /Hyperlipidemia/ PTB / BA /COPD/Drug intake

Personal History

Smoking / Alcohol / Pan chewing

Menstrual /Obstetric history

Family History : DM/ HTN /CAD /Hyperlipidemia

GENERAL EXAMINATION

VITALS : Pulse BP RR Temp

Fundus

CVS : JVP S1 S2 S3 S4 Murmurs

RS : **ABDOMEN**: **CNS**:

INVESTIGATIONS

Hb TC DC: P L E M ESR

Glucose Urea Creatinine

Lipid Profile..Total Cholesterol LDL VLDL HDL TGL

CREATINE KINASE …TOTAL CK MB

ECG in all leads

ECHOCARDIOGRAPHY

M Mode : TAM

2 D ECHO

(1) Tricuspid Annular Fractional Shortening

(2) RV Dimensions (cm) Basal Base to Apex

(3) RV (cm2) Diastolic Area Systolic Area

Fractional Area Change

(4) RV Wall Motion (N / Hypo / akinetic / dyskinetic)

(5) RV Wall Thickness (cm)

(6) IVS Paradoxical Motion

(7) Valves---TV MV PV AV

(8) Other Chambers RA LA LV

(9)Pericardial Effusion

PULSED WAVE DOPPLER ECHO

E ; A (m/s); E/A ;

E wave deceleration time (ms)

DOPPLER TISSUE IMAGING

(1)TAV (cm/s) - Peak Systolic (Sm) Early Diastolic (Em)

Late Diastolic (Am)

(2) IVS (cm/s) - Sm Em Am

(3) MPI = IVCT + IVRT / ET

[IVCT(ms) ,IVRT(ms) , ET]

APPENDIX II - MASTER CHART

MASTER CHART : CLINICAL PROFILE

S.No:	AGE	SEX	SYMPTOMS	KILLIPS CLASS	PAST HISTORY	RISK FACTORS	CBC	RBS	RFT	CHOLESTEROL	CK TOTAL	CK-MB	ECG
1	70	M	1,6	I	-	1	ANEMIA	152	NORMAL	143	310	37	IW+RV+PW
2	44	M	1,6,9	I	1,2	1	NORMAL	439	NORMAL	162	240	36	IW+RV+PW
3	65	F	1,3,6,8	I	-	4	ANEMIA	180	NORMAL	126	90	10	IW+RV
4	58	F	3,8	II		3,4	ANEMIA	108	NORMAL	122	414	48	IW+RV
5	55	F	1,2,6,8	II	2	4	ANEMIA	245	NORMAL	192	840	174	IW+RV
6	52	M	1,2,6,9	I	-	1	ANEMIA	119	NORMAL	144	210	32	IW+RV
7	68	M	1,2,3,6,9	I	-	-	NORMAL	65	NORMAL	120	420	60	IW+RV
8	33	F	1,6,8,9	I	2	-	ANEMIA	240	NORMAL	210	450	81	IW+RV+PW
9	63	F	1,6,9	I	1,2	4	ANEMIA	312	NORMAL	214	96	21	IW+RV+PW
10	63	M	1,2,3,6,9	I	-	1,2	NORMAL	357	NORMAL	171	900	159	IW+PW
11	60	M	1,3,6,8	I	-	1,2	NORMAL	109	NORMAL	138	317	41	IW+RV+PW
12	48	M	1,6,9	I	-	1	ANEMIA	74	NORMAL	194	1298	168	IW+RV
13	55	M	1	I	1	2	NORMAL	199	NORMAL	190	76	10	IW+RV
14	30	M	1,6	I	-	1,2	NORMAL	137	NORMAL	140	218	36	IW+RV
15	43	M	1	I	1	1,2	NORMAL	124	NORMAL	120	292	22	IW+RV
16	59	M	1	I	-	1,2	ANEMIA	64	NORMAL	140	198	44	IW+RV+PW
17	86	M	1,3,6,8,9	I	-	-	ANEMIA	122	NORMAL	126	90	18	IW+RV
18	57	M	1,6,9	IV	-	1	NORMAL	88	NORMAL	120	198	27	IW+RV
19	40	M	1,6,8,9	I	-	1,2	NORMAL	78	NORMAL	110	123	59	IW+RV
20	58	M	1	I	1	1,2	NORMAL	100	NORMAL	122	444	44	IW+RV
21	35	M	1,2,6	I	2	-	NORMAL	193	NORMAL	154	302	23	IW+RV
22	28	M	1	I	-	1,2	NORMAL	372	NORMAL	218	68	16	IW+RV+PW
23	60	F	1,2,6,9	IV	-	1	ANEMIA	94	NORMAL	122	77	17	IW+RV+PW
24	63	M	1,6,8,9	II	1	1	NORMAL	177	NORMAL	140	21	6	IW+RV
25	60	M	1,3,6,9	IV	1,3	1	ANEMIA	69	NORMAL	186	91	39	IW+RV+PW
26	48	M	1,2,4,9	I	1	1	NORMAL	87	NORMAL	123	437	32	IW
27	52	M	1,3,6,8,9	I	-	-	NORMAL	105	NORMAL	160	953	240	IW+RV+PW
28	55	M	1	I	-	1	ANEMIA	90	NORMAL	120	124	16	IW+RV
29	60	M	1	I	1	1	NORMAL	110	NORMAL	126	148	42	IW
30	58	M	1,2,9,10	II	2	-	ANEMIA	307	NORMAL	210	229	33	IW+RV
31	66	M	1,6	I	-	1	ANEMIA	94	NORMAL	120	285	44	IW+RV+PW
32	56	M	1,3,9	I	-	-	ANEMIA	272	NORMAL	180	133	13	IW+RV
33	45	F	1,2	I	1,2	4	NORMAL	521	NORMAL	270	23	12	IW+RV
34	55	M	1,2	II	2	-	ANEMIA	312	NORMAL	210	102	22	IW+RV+PW
35	50	M	1,2	I	1	1,2	NORMAL	78	NORMAL	110	102	20	IW+RV+PW

Keys to Master Chart

Symptoms :

1=Chest Pain; 2=Dyspnea ; 3=Giddiness ; 4=Fatigue ; 5=Epigastric Pain ; 6=Diaphoresis ;7= Syncope ; 8=Vomiting ; 9=Palpitation ; 10= Pedal Edema.

Past History :

1 = Systemic Hypertension; 2 = Diabetes Mellitus .

Risk Factors :

1 = Smoking ; 2 = Alcoholism ;

3 = Tobacco Chewing ; 4 = Post menopausal.

ECHOCARDIOGRAPHIC EVALUATION OF RIGHT VENTRICLE (Group A)

S.No:	TAM mm	TAFS	RV BASE cm	RV L A cm	RVEDA cm2	RVESA cm2	RVFAC	RV WT mm	RV W M	IVS PM	E ms	A ms	E/A	E wave DT ms
1	15	23.33	3	7.9	16.68	12.42	25.53	7	HYPO	NO	0.56	0.46	1.24	289
2	11	26.66	2.9	8.7	19.33	13	32.74	4	HYPO	NO	0.62	0.40	1.87	267
3	15	33.33	3.2	7.7	10.4	5.94	42.88	4	HYPO	NO	0.48	0.39	1.34	214
4	16	20	3	7	15	12.1	19.33	4	HYPO	NO	0.52	0.46	1.33	257
5	20	18.75	3.4	7	14.64	11.22	23.36	4	HYPO	NO	0.56	0.44	1.47	282
6	15	20.68	3.8	6.8	15.36	7.82	49.08	4	HYPO	NO	0.52	0.48	1.26	257
7	15	33.33	4.4	6.2	13	8.48	34.76	4	AKINETIC	NO	0.73	0.48	1.82	187
8	20	12.5	2.7	6.1	13.15	7.3	44.48	3	HYPO	NO	0.58	0.68	0.85	195
9	14	25	3	7.5	14.2	9.1	35.91	5	HYPO	NO	0.72	0.42	1.16	240
10	15	16.66	3.2	7.8	16.2	10.3	36.41	5	HYPO	NO	0.52	0.48	1.36	280
11	19	20	4.1	7.4	16.35	8.49	48.07	5	NORMAL	NO	0.72	0.45	1.6	288
12	20	41.66	3.4	6.2	15.42	12.17	21.07	3	HYPO	NO	0.74	0.46	1.5	225
13	23	17.85	3.5	7.7	12.26	5.41	55.87	4	AKINETIC	NO	0.64	0.46	1.23	242
14	16	11.11	3.3	7.6	13.1	8.3	36.64	5	AKINETIC	NO	0.62	0.48	0.87	290
15	18	30	3.8	7.5	16.25	9.8	39.69	4	HYPO	NO	0.50	0.46	1.29	280
16	22	33	3.3	7.2	14.25	9.3	34.73	4	HYPO	NO	0.50	0.36	1.38	288
17	20	12	2.4	6.5	11.98	8.97	25.12	5	HYPO	NO	0.48	0.36	1.69	269
18	17	27.27	3.5	7.6	14.3	10.3	27.97	4	HYPO	NO	0.52	0.43	1.29	192
19	16	29.16	3.3	7.8	13.9	8.8	36.69	4	HYPO	NO	0.54	0.42	1.27	198
20	15	26.82	3	7	14.99	10.01	33.22	4	NORMAL	NO	0.50	0.42	1.19	197
21	16	23.33	2.9	7.9	13.25	9.07	31.54	4	HYPO	NO	0.49	0.50	0.98	291
22	15	30.3	2.8	8.2	12.79	8.09	36.74	3	NORMAL	NO	0.54	0.39	1.86	278
23	18	39.47	3.6	6.1	10.81	4.86	55.04	5	HYPO	NO	0.53	0.35	1.51	225
24	14	29.72	3.1	7.9	14.4	8.79	38.95	6	HYPO	NO	0.52	0.36	0.78	257
25	12	28.5	3.2	7.9	11.31	4.82	57.38	5	HYPO	NO	0.42	0.41	0.85	262
26	14	42.22	4.1	7.7	23.67	14.01	40.81	4	HYPO	NO	0.56	0.49	1.14	242
27	16	17.14	2.8	6.5	11.72	7.05	39.84	4	HYPO	NO	0.42	0.34	2.28	325
28	16	42.1	4.2	8.9	18.38	7.74	57.88	4	HYPO	NO	0.37	0.44	0.61	286
29	24	4	3	6	17.64	10.64	39.68	6	NORMAL	NO	0.50	0.24	1.66	264
30	13	12.12	3.9	7	11.7	7	40.17	4	HYPO	NO	0.50	0.38	1.20	280
31	12	33.33	3.5	6.8	12.68	8.94	29.49	5	NORMAL	NO	0.52	0.39	1.41	293
32	10	33.33	2.6	5.2	11.98	7.69	35.8	4	NORMAL	NO	0.49	0.33	1.40	193
33	10	57.14	2.9	6.4	16.19	7.33	54.72	4	HYPO	NO	0.57	0.33	1.72	185
34	20	23.3	2.6	6.4	12.29	5.97	51.42	5	AKINETIC	NO	0.48	0.36	1.49	200
35	20	7.69	3.7	7.2	13.23	10.21	22.82	4	HYPO	NO	0.48	0.36	1.60	232

ECHOCARDIOGRAPHIC EVALUATION OF RV (Contd..) (Group A)

S.No:	IVCT (ms)	IVRT (ms)	ET (ms)	MPI	TAV Sm (cm/s)	TAV Em (cm/s)	TAV Am (cm/s)	IVS Sm (cm/s)	IVS Em (cm/s)	IVS Am (cm/s)
1	82	146	217	1.05	10	10	15	9	9	12
2	82	107	274	0.68	11	12	19	8	12	9
3	80	140	210	1.04	16	12	9	6	6	8
4	42	70	190	0.58	12	9	8	10	6	7
5	28	85	260	0.43	12	9	13	8	8	11
6	25	100	306	0.4	8	12	8	6	10	10
7	18	128	128	1.14	9	6	7	6	6	7
8	36	36	250	0.28	14	22	13	8	13	10
9	40	52	240	0.38	14	14	10	9	8	9
10	62	80	242	0.58	10	9	8	8	6	7
11	57	117	217	1.26	15	13	14	7	8	9
12	43	82	238	0.52	14	13	10	11	10	9
13	21	80	196	0.51	13	11	12	7	7	8
14	60	50	196	0.56	10	9	7	8	6	8
15	45	86	220	0.59	14	10	8	10	8	9
16	60	80	220	0.63	11	10	9	8	6	6
17	50	75	338	0.36	12	9	20	5	4	10
18	55	80	220	0.61	14	11	10	8	6	6
19	50	60	200	0.55	13	10	9	8	6	7
20	61	43	221	0.47	8	6	10	5	4	7
21	85	139	263	0.85	11	9	9	10	10	9
22	78	36	224	0.51	11	9	13	6	5	7
23	57	75	214	0.62	9	10	12	10	11	13
24	46	57	238	0.43	11	9	10	8	7	9
25	39	128	238	0.7	10	7	11	5	7	8
26	32	50	256	0.32	14	15	41	7	9	6
27	18	32	349	0.14	12	10	12	6	7	8
28	85	43	256	0.5	14	18	22	8	12	9
29	36	260	128	0.63	7	4	6	6	6	4
30	78	64	249	0.57	11	9	16	6	4	8
31	57	39	238	0.4	7	6	6	6	6	5
32	28	192	199	1.1	10	6	12	5	6	7
33	61	85	246	0.59	11	9	13	6	6	10
34	85	40	250	0.5	14	17	20	8	12	9
35	75	107	278	0.65	10	8	8	9	7	10

ECHOCARDIOGRAPHIC EVALUATION OF RV (CONTROLS)

S.No:	TAM mm	TAFS	RV BASE cm	RV L A cm	RVEDA cm2	RVESA cm2	RVFAC	RV WTmm	RV W M	IVS PM	E ms	A ms	E/A	E wave DT ms
1	24	15.4	2.4	7.4	16	8	50	0.36	NORMAL	NO	0.46	0.33	1.17	160
2	22	16.2	2.4	7.6	18	9	50	0.4	NORMAL	NO	0.48	0.33	1.23	168
3	22	14.4	2.6	7.8	18	10	44.44	0.4	NORMAL	NO	0.49	0.37	1.12	160
4	24	16.2	2.2	7.4	18	10	44.44	0.4	NORMAL	NO	0.52	0.39	1.13	170
5	22	14.8	2.8	7.8	17	9	47.05	0.4	NORMAL	NO	0.5	0.37	1.15	172
6	24	15.2	2.4	7.6	18	9	50	0.3	NORMAL	NO	0.48	0.35	1.16	180
7	23	14.4	2.4	7.8	18	9	50	0.4	NORMAL	NO	0.46	0.35	1.11	192
8	25	16.2	2.6	7.4	16	8	50	0.4	NORMAL	NO	0.48	0.37	1.10	168
9	24	14.8	2.8	7.5	17	9	47.05	0.4	NORMAL	NO	0.49	0.37	1.12	172
10	24	16	2.2	7.4	15	8	46.66	0.3	NORMAL	NO	0.5	0.37	1.15	168

Sl.No:	IVCT ms	IVRT ms	ET ms	MPI	TAV Sm	TAV Em	TAV Am	IVS Sm	IVS Em	IVS Am
1	44	36	320	0.25	14	14	14.4	9.2	11.2	12
2	42	34	320	0.23	14.2	13	14	9	11.4	11.6
3	42	34	360	0.21	13.8	13	13.8	9.4	11.2	11.4
4	44	36	340	0.235	14.4	14.2	14	8.8	11	11.2
5	43	33	344	0.22	13.8	13.6	14.2	9	11.4	11.6
6	42	34	360	0.21	14.2	14	14.4	10	11	12
7	40	32	362	0.19	14.6	13.4	13.8	10	11	12
8	42	38	348	0.22	14.2	13.6	13.8	9.8	11	12
9	42	36	344	0.22	14.4	13.8	14	10	11.2	12
10	40	34	340	0.21	14.4	13.8	14.2	10	11	12